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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2	Apr 08	"Ask CAS" for self-help around the clock
NEWS	3	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS	4	Apr 09	ZDB will be removed from STN
NEWS	5	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS	6	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS	7	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS	8	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS	9	Jun 03	New e-mail delivery for search results now available
NEWS	10	Jun 10	MEDLINE Reload
NEWS	11	Jun 10	PCTFULL has been reloaded
NEWS	12	Jul 02	FOREGE no longer contains STANDARDS file segment
NEWS	13	Jul 22	USAN to be reloaded July 28, 2002; saved answer sets no longer valid
NEWS	14	Jul 29	Enhanced polymer searching in REGISTRY
NEWS	15	Jul 30	NETFIRST to be removed from STN
NEWS	16	Aug 08	CANCERLIT reload
NEWS	17	Aug 08	PHARMAMarketLetter(PHARMAML) - new on STN
NEWS	18	Aug 08	NTIS has been reloaded and enhanced
NEWS	19	Aug 19	Aquatic Toxicity Information Retrieval (AQUIRE) now available on STN
NEWS	20	Aug 19	IFIPAT, IFICDB, and IFIUDB have been reloaded
NEWS	21	Aug 19	The MEDLINE file segment of TOXCENTER has been reloaded
NEWS	22	Aug 26	Sequence searching in REGISTRY enhanced
NEWS	23	Sep 03	JAPIO has been reloaded and enhanced
NEWS	24	Sep 16	Experimental properties added to the REGISTRY file
NEWS	25	Sep 16	CA Section Thesaurus available in CAPLUS and CA
NEWS	26	Oct 01	CASREACT Enriched with Reactions from 1907 to 1985
NEWS	27	Oct 21	EVENTLINE has been reloaded
NEWS	28	Oct 24	BEILSTEIN adds new search fields
NEWS	29	Oct 24	Nutraceuticals International (NUTRACEUT) now available on STN
NEWS	30	Oct 25	MEDLINE SDI run of October 8, 2002
NEWS	31	Nov 18	DKILIT has been renamed APOLLIT
NEWS	32	Nov 25	More calculated properties added to REGISTRY
NEWS	33	Dec 02	TIBKAT will be removed from STN
NEWS	34	Dec 04	CSA files on STN
NEWS	35	Dec 17	PCTFULL now covers WP/PCT Applications from 1978 to date
NEWS	36	Dec 17	TOXCENTER enhanced with additional content
NEWS	37	Dec 17	Adis Clinical Trials Insight now available on STN
NEWS	38	Dec 30	ISMEC no longer available
NEWS	39	Jan 13	Indexing added to some pre-1967 records in CA/CAPLUS
NEWS	40	Jan 21	NUTRACEUT offering one free connect hour in February 2003
NEWS	41	Jan 21	PHARMAML offering one free connect hour in February 2003
NEWS	42	Jan 29	Simultaneous left and right truncation added to COMPENDEX, ENERGY, INSPEC
NEWS	43	Feb 13	CANCERLIT is no longer being updated
NEWS	44	Feb 24	METADEX enhancements
NEWS	45	Feb 24	PCTGEN now available on STN
NEWS	46	Feb 24	TEMA now available on STN

NEWS 47 Feb 26 NTIS now allows simultaneous left and right truncation
NEWS 48 Feb 26 PCTFULL now contains images
NEWS 49 Mar 04 SDI PACKAGE for monthly delivery of multifile SDI results

NEWS EXPRESS January 6 CURRENT WINDOWS VERSION IS V6.01a,
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FILE 'HOME' ENTERED AT 16:04:17 ON 04 MAR 2003

=> file uspatfull

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FILE 'USPATFULL' ENTERED AT 16:04:30 ON 04 MAR 2003

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FILE COVERS 1971 TO PATENT PUBLICATION DATE: 4 Mar 2003 (20030304/PD)
FILE LAST UPDATED: 4 Mar 2003 (20030304/ED)
HIGHEST GRANTED PATENT NUMBER: US6530088
HIGHEST APPLICATION PUBLICATION NUMBER: US2003041363
CA INDEXING IS CURRENT THROUGH 4 Mar 2003 (20030304/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 4 Mar 2003 (20030304/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Dec 2002
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Dec 2002

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>>> publications, starting in 2001, for the inventions covered in <<<
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This file contains CAS Registry Numbers for easy and accurate
substance identification.

=> s stem cells and nitric oxide
126332 STEM
319013 CELLS
9569 STEM CELLS
(STEM(W)CELLS)
67061 NITRIC
475839 OXIDE
6927 NITRIC OXIDE
(NITRIC(W)OXIDE)
L1 507 STEM CELLS AND NITRIC OXIDE

=> s l1 and hematopoiesis
2016 HEMATOPOIESIS
L2 189 L1 AND HEMATOPOIESIS

=> s l2 and nitric oxide synthase
67061 NITRIC
475839 OXIDE
9133 SYNTHASE
1387 NITRIC OXIDE SYNTHASE
(NITRIC(W)OXIDE(W)SYNTHASE)
L3 142 L2 AND NITRIC OXIDE SYNTHASE

=> s l3 and pd<1999
2434829 PD<1999
(PD<19990000)
L4 0 L3 AND PD<1999

=> s l3 and pd<2000
2604557 PD<2000
(PD<20000000)
L5 2 L3 AND PD<2000

=> d l2 1-2

L2 ANSWER 1 OF 189 USPATFULL
AN 2003:57533 USPATFULL
TI Serpin polynucleotides, polypeptides, and antibodies
IN Ruben, Steven M., Olney, MD, UNITED STATES
Ni, Jian, Germantown, MD, UNITED STATES
PA Human Genome Sciences, Inc., Rockville, MD, UNITED STATES, 20850 (U.S.
corporation)
PI US 2003040097 A1 20030227
AI US 2002-116166 A1 20020405 (10)
RLI Continuation of Ser. No. US 2000-641721, filed on 21 Aug 2000, PENDING
Continuation-in-part of Ser. No. WO 2000-US5082, filed on 29 Feb 2000,
UNKNOWN
PRAI US 1999-122276P 19990301 (60)
US 1999-124094P 19990312 (60)
US 1999-149452P 19990818 (60)
DT Utility
FS APPLICATION
LN.CNT 8865
INCL INCLM: 435/226.000
INCLS: 435/320.100; 435/325.000; 435/069.100; 536/023.200; 435/184.000

NCL NCLM: 435/226.000
 NCLS: 435/320.100; 435/325.000; 435/069.100; 536/023.200; 435/184.000
 IC [7]
 ICM: C12N009-64
 ICS: C12N009-99; C07H021-04; C12P021-02; C12N005-06

 L2 ANSWER 2 OF 189 USPATFULL
 AN 2003:57524 USPATFULL
 TI Secreted protein HT5GJ57
 IN Ruben, Steven M., Olney, MD, UNITED STATES
 Komatsoulis, George, Silver Spring, MD, UNITED STATES
 Duan, Roxanne D., Bethesda, MD, UNITED STATES
 Rosen, Craig A., Laytonsville, MD, UNITED STATES
 Moore, Paul A., Germantown, MD, UNITED STATES
 Shi, Yanggu, Gaithersburg, MD, UNITED STATES
 LaFleur, David W., Washington, DC, UNITED STATES
 Ebner, Reinhard, Gaithersburg, MD, UNITED STATES
 Olsen, Henrik S., Gaithersburg, MD, UNITED STATES
 Brewer, Laurie A., St. Paul, MN, UNITED STATES
 Florence, Kimberly A., Rockville, MD, UNITED STATES
 Young, Paul E., Gaithersburg, MD, UNITED STATES
 Mucenski, Michael, Cincinnati, OH, UNITED STATES
 Endress, Gregory A., Florence, MA, UNITED STATES
 Soppet, Daniel R., Centreville, VA, UNITED STATES
 PA Human Genome Sciences, Inc., Rockville, MD, UNITED STATES, 20850 (U.S. corporation)
 PI US 2003040088 A1 20030227
 AI US 2001-984271 A1 20011029 (9)
 RLI Division of Ser. No. US 2000-482273, filed on 13 Jan 2000, PENDING
 Continuation-in-part of Ser. No. WO 1999-US15849, filed on 14 Jul 1999, UNKNOWN
 PRAI US 1998-92921P 19980715 (60)
 US 1998-92922P 19980715 (60)
 US 1998-92956P 19980715 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 24720
 INCL INCLM: 435/183.000
 INCLS: 435/006.000; 435/069.100; 435/325.000; 435/320.100; 530/350.000; 536/023.200
 NCL NCLM: 435/183.000
 NCLS: 435/006.000; 435/069.100; 435/325.000; 435/320.100; 530/350.000; 536/023.200
 IC [7]
 ICM: C12Q001-68
 ICS: C07H021-04; C12N009-00; C12P021-02; C12N005-06

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(FILE 'HOME' ENTERED AT 16:04:17 ON 04 MAR 2003)

FILE 'USPATFULL' ENTERED AT 16:04:30 ON 04 MAR 2003

L1 507 S STEM CELLS AND NITRIC OXIDE
 L2 189 S L1 AND HEMATOPOIESIS
 L3 142 S L2 AND NITRIC OXIDE SYNTHASE
 L4 0 S L3 AND PD<1999
 L5 2 S L3 AND PD<2000

=> d l5 1-2 bib, kwic

L5 ANSWER 1 OF 2 USPATFULL

AN 1999:141294 USPATFULL
 TI Methods for enhancing angiogenesis with endothelial progenitor cells
 IN Isner, Jeffrey M., Weston, MA, United States
 Asahara, Takayuki, Arlington, MA, United States
 PA St. Elizabeth's Medical Center of Boston, Boston, MA, United States
 (U.S. corporation)
 PI US 5980887 19991109 <--
 AI US 1996-744882 19961108 (8)
 DT Utility
 FS Granted
 EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: Gambel,
 Phillip
 LREP Conlin, David G., Resnick, David S. Dike, Bronstein, Roberts & Cushman,
 LLP
 CLMN Number of Claims: 11
 ECL Exemplary Claim: 1
 DRWN 43 Drawing Figure(s); 7 Drawing Page(s)
 LN.CNT 1104
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.
 PI US 5980887 19991109 <--
 DETD . . . typically begins as a cluster formation, or blood island,
 comprised of EC progenitors (e.g. angioblasts) at the periphery and
 hematopoietic **stem cells** (HSCs) at the center (3).
 In addition to this intimate and predictable spatial association, such
 EC progenitors and HSCs share. . .
 DETD The demonstration that transplants of HSCs derived from peripheral blood
 can provide sustained hematopoietic recovery constitutes inferential
 evidence for circulating **stem cells**. (5). This
 observation is now being exploited clinically as an alternative to bone
 marrow transplantation.
 DETD . . . genes or their encoded gene products to enhance the activity of
 targeted cells, while simultaneously inducing angiogenesis, including,
 for example, **nitric oxide synthase**,
 L-arginine, fibronectin, urokinase, plasminogen activator and heparin.
 DETD ECs uniquely express endothelial constitutive **nitric**
oxide synthase (ecNOS). Accordingly, MB.sup.CD34+,
 MB.sup.CD34- and AT.sup.CD34+ were investigated for expression of
 ecNOS by RT-PCR (15). ecNOS mRNA was not detectable. . . however,
 ecNOS mRNA was markedly increased (FIG. 5). Functional evidence of ecNOS
 protein in AT.sup.CD34+ was documented by measurement of **nitric**
oxide in response to the EC-dependent agonist, acetylcholine
 (Ach), and the EC-specific mitogen, vascular endothelial growth factor
 (VEGF) (16) (FIG. 5); . . .
 DETD Cell-cell interaction is considered to play a decisive role in cell
 signaling, differentiation, and proliferation during
hematopoiesis (19) and angiogenesis (20). To study the impact of
 MB.sup.CD34+ interaction with mature ECs on the differentiation of
 MB.sup.CD34+ into. . .

 L5 ANSWER 2 OF 2 USPATFULL
 AN 1999:137330 USPATFULL
 TI Therapeutic uses for **nitric oxide** inhibitors
 IN Enikolopov, Grigori N., Cold Spring Harbor, NY, United States
 Peunova, Natalia I., Cold Spring Harbor, NY, United States
 Kuzin, Boris A., Moscow, Russian Federation
 Michurina, Tatiyana, Moscow, Russian Federation
 PA Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, United States
 (U.S. corporation)
 PI US 5977181 19991102 <--
 AI US 1997-969475 19971113 (8)
 PRAI US 1996-30690P 19961113 (60)
 US 1997-45411P 19970502 (60)

DT Utility
FS Granted
EXNAM Primary Examiner: Criares, Theodore J.
LREP Hamilton, Brook, Smith & Reynolds, P.C.
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1655

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

TI Therapeutic uses for **nitric oxide** inhibitors

PI US 5977181 19991102

<--

AB The present invention is based on the discovery that **nitric oxide** (NO) is an important growth regulator in an intact developing organism. In particular, the present invention relates to a method of increasing in a mammal a population of hematopoietic **stem cells** in bone marrow which are capable of undergoing normal **hematopoiesis** and differentiation, wherein the bone marrow is contacted with an inhibitor of NO, such as an inhibitor of **nitric oxide synthase** (NOS), thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation. The present invention also relates to a method of increasing a population of cells in S phase in.

SUMM The present invention is based on the discovery that **nitric oxide** (NO) is an important growth regulator in an intact developing organism. In particular, the present invention relates to a method of increasing in a mammal a population of hematopoietic **stem cells**, including precursors to myeloid, lymphoid and erythroid cells, in bone marrow which are capable of undergoing normal **hematopoiesis** and differentiation, wherein the bone marrow is contacted with an inhibitor of NO, such as an inhibitor of **nitric oxide synthase** (NOS), thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation. The method can be carried out in vivo or ex vivo. In addition, the method can be used.

SUMM The present invention also relates to a method for treating a mammal to increase a population of hematopoietic **stem cells** in bone marrow of the mammal which are capable of undergoing normal **hematopoiesis** and differentiation. In the method, the bone marrow of the mammal is contacted with an inhibitor of NOS, thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation. The method can further comprise contacting the bone marrow with at least one agent which induces differentiation of.

SUMM In one embodiment of the method for treating a mammal to increase a population of hematopoietic **stem cells** in bone marrow of the mammal which are capable of undergoing normal **hematopoiesis** and differentiation, bone marrow which is to be transplanted is obtained, wherein the bone marrow to be transplanted can be. . . is transplanted into the mammal being treated, thereby providing the mammal with bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation. The method can further comprise treating the mammal with an inhibitor of NOS before or after transplanting the.

SUMM . . . increasing a population of dividing cells in a tissue of a mammal comprising contacting the cells with an inhibitor of **nitric oxide**. In one embodiment, the present invention also relates to a method of increasing a population of cells in S phase.

SUMM . . . thereby inhibiting differentiation and inducing proliferation of cells of the tissue, then contacting the selected tissue with a compound (e.g., **nitric oxide**, a growth factor or a combination of both) which inhibits proliferation and induced differentiation. In one embodiment, the method involves. . .

SUMM . . . method, bone marrow is contacted with an inhibitor of NOS, thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation; and at least one agent (e.g., a hematopoietic growth factor) selected to induce specific differentiation of the hematopoietic. . .

SUMM Results of the work described herein have shown that a transcellular messenger (**nitric oxide** (NO)) plays a critical role in tissue differentiation and organism development. NO regulates the balance between cell proliferation and cell. . .

SUMM Accordingly, the present invention relates to a method of increasing in a mammal a population of hematopoietic **stem cells**, including precursors to myeloid, lymphoid and erythroid cells, in bone marrow which are capable of undergoing normal **hematopoiesis** and differentiation, by contacting the bone marrow with an inhibitor of NO, such as an inhibitor of NOS. The present invention includes a method for treating a mammal to increase a population of hematopoietic **stem cells** in bone marrow of the mammal which are capable of undergoing normal **hematopoiesis** and differentiation, in which the bone marrow of the mammal is contacted with an inhibitor of NOS.

SUMM . . . increasing a population of dividing cells in a tissue of a mammal comprising contacting the cells with an inhibitor of **nitric oxide**. In one embodiment, the present invention can also be used to increase a population of cells (targeted cells) in S. . .

SUMM . . . lymphocytes, neutrophils and platelets). For example, in the embodiment wherein a mammal is treated to increase a population of hematopoietic **stem cells** in the bone marrow of the mammal by contacting the bone marrow of the mammal with an inhibitor of NOS,. . .

SUMM . . . balance between cell proliferation and cell differentiation. Moreover, results shown here demonstrate that NO acts as a crucial regulator of **hematopoiesis** after bone marrow (BM) transplantation. NO regulates the maturation of both the erythroid and myeloid lineages. These data demonstrate that manipulations of NOS activity and NO levels during **hematopoiesis** can be used to alter (enhance or reduce) blood cell production. This is useful for preventive and therapeutic intervention.

SUMM As also described herein, the role of NO in **hematopoiesis** was examined. To demonstrate the presence of NOS in the bone marrow (BM) cells, BM from adult mice was tested. . .

SUMM A mouse model of syngeneic BM transfer was used to evaluate the role of NO in **hematopoiesis**. Mice were irradiated to inhibit **hematopoiesis** in the recipient animal, BM was transplanted from syngeneic animals, and the animals were treated with specific NOS inhibitors. This procedure permits the proliferation, differentiation and survival of only the transplanted cells. To study the changes in **hematopoiesis** introduced by NOS inhibitors, the colonies in the spleen were monitored to test the differentiation of erythroid cells, and the. . . of the recipients were monitored to test the differentiation of cells of the granulocyte-macrophage lineage. The role of NO on **hematopoiesis** was tested by injecting the animals with the specific and structurally unrelated NOS inhibitors L-nitroarginine methyl ester (L-NAME), and 2-ethyl-2-thiopseudourea. . .

SUMM Taken together, the results of these studies indicate that NO modulates **hematopoiesis** after BM transplantation. This confirms the role of NO as a major regulatory factor in the organism controlling the balance. . . .

SUMM The results of work described herein support the ability of NO to act as a crucial regulator of **hematopoiesis** after bone marrow transplantation (BMT). NO regulates maturation of both erythroid and myeloid cell lineages. By interfering with NO production. . . . and 20-fold for the erythroid lineage. The data described herein demonstrates that manipulations of NOS activity and NO levels during **hematopoiesis** can be used for therapeutic purposes to influence self renewal and differentiation of hematopoietic **stem cells**, and to replace damaged or defective cells. Areas of application include enhancement of blood cell and myeloid cell formation following. . . .

SUMM . . . chondrocyte differentiation. These results show that manipulation of NO production can regulate growth and differentiation of osteoblasts, chondrocytes, or mesenchymal **stem cells**. This can be used for amplification and further differentiation of cells in the injured tissue, or for cell implants (in. . . .

DETD **Nitric Oxide Regulates Cell Proliferation During Drosophila Development**

DETD **Nitric Oxide Regulates Hematopoiesis in Animals Erythroid Differentiation**

DETD . . . cGy total body irradiation within 3-4 hours before transplantation. This dosage was tested to be enough for complete suppression of **hematopoiesis** in the irradiated recipient animals. BM cells were flushed from the femurs of syngeneic donors and injected intravenously (10.sup.5 BM. . . .

DETD . . . granulocyte colony stimulating factor (G-CSF-R) and erythropoietin (EpoR). The appearance of each of these receptors marks a specific stage in **hematopoiesis**.

DETD **Stem Cells in the Bone Marrow**

DETD . . . the presence of various growth factor receptors which serve as markers of the differentiation stage and indicate the presence of **stem cells** and multipotent precursor cells. The BM preparations were tested for cells expressing receptors to HSF (ligand of c-kit), GM-CSF, G-CSF. . . . number of c-kit-positive and IL-3-R-positive cells, suggesting that the population of cells in the BM becomes highly enriched in hematopoietic **stem cells**. At the same time the number of cells expressing receptors for G-CSF, which marks the later stages of differentiation, decreases almost three-fold, while the number of GM-CSF-R-positive cells is slightly decreased. This suggests that inhibition of NOS during **hematopoiesis** selectively enriches the BM in undifferentiated **stem cells** which have already acquired c-kit and IL3 receptors, but have not proceeded to the later stages when the receptor for. . . .

DETD The critical question is whether undifferentiated **stem cells** which accumulate in the bone marrow as a result of treatment with NOS inhibitors have the capacity to revert to normal state and resume normal **hematopoiesis** process once the action of NOS inhibitors is suspended. The failure to do so might indicate that the cells become. . . . with NOS inhibitors was halted 7-9 days after the BM transfer and checked the BM cells for the presence of **hematopoiesis** markers 1-7 days after termination of injections. Control mice continued to receive the daily injections, The results (Table 4) demonstrate. . . . cells were able to resume their differentiation and to proceed to the later stages normally. This indicates that enrichment in **stem cells** after treatment with NOS inhibitors is reversible and can be used to "boost" the number of **stem cells** before inducing them to

proceed further along their differentiation pathways.

DETD **Nitric Oxide** Regulates Brain Development In Vertebrates

DETD It has been recently demonstrated that **nitric oxide** (NO), a multifunctional second messenger, is involved in cell and tissue differentiation and organism development. NO synthase (NOS) controls the. . . as a result, regulates the balance between cell proliferation and differentiation in cultured neuronal cells, in developing *Drosophila*, and during **hematopoiesis** in mammals (Peunova et al., 1996; Kuzin et al., 1996; Michurina et al., 1997). Here, whether NOS is involved in. . .

CLM What is claimed is:

1. A method of increasing in a mammal a population of hematopoietic **stem cells** in bone marrow which are capable of undergoing normal **hematopoiesis** and differentiation, comprising contacting the bone marrow with an inhibitor of **nitric oxide synthase**, thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation.

3. A method according to claim 2 further comprising implanting the bone marrow having an increased population of hematopoietic **stem cells** into a mammal in need thereof.

7. A method according to claim 1 wherein the inhibitor of **nitric oxide synthase** is selected from the group consisting of L-nitroarginine methyl ester, 2-ethyl-2-thiopseudourea, aminoguanidine hemisulfate and N-monomethyl-L-arginine.

8. A method for treating a mammal to increase a population of hematopoietic **stem cells** in bone marrow of the mammal which are capable of undergoing normal **hematopoiesis** and differentiation, comprising contacting the bone marrow of the mammal with an inhibitor of **nitric oxide synthase**, thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation.

10. A method for treating a mammal to increase a population of hematopoietic **stem cells** in bone marrow of the mammal which are capable of undergoing normal **hematopoiesis** and differentiation, comprising the steps of: a) obtaining bone marrow which is to be transplanted into the mammal; b) contacting the bone marrow to be transplanted with an inhibitor of **nitric oxide synthase**; c) transplanting the bone marrow of step (b) into the mammal to be treated, thereby providing the mammal with bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation.

11. A method according to claim 10 further comprising: d) treating the mammal with an enhancer of **nitric oxide synthase** after transplanting the bone marrow.

12. A method according to claim 10 further comprising: d) treating the mammal with an inhibitor of **nitric oxide synthase** after transplanting the bone marrow.

. . . method of producing a subpopulation of hematopoietic cells comprising the steps of: a) contacting bone marrow with an inhibitor of **nitric oxide synthase**, thereby producing

bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation; and b) contacting the bone marrow with at least one hematopoietic growth factor selected to induce specific differentiation. . .

=> s stem cell? and nitric oxide synthase and hematope?

126332 STEM
641162 CELL?
11080 STEM CELL?
(STEM(W) CELL?)
67061 NITRIC
475839 OXIDE
9133 SYNTHASE
1387 NITRIC OXIDE SYNTHASE
(NITRIC(W) OXIDE(W) SYNTHASE)
4 HEMATOPE?

L6 0 STEM CELL? AND NITRIC OXIDE SYNTHASE AND HEMATOPE?

=> s stem cell? and nitric oxide synthase and hematopo?

126332 STEM
641162 CELL?
11080 STEM CELL?
(STEM(W) CELL?)
67061 NITRIC
475839 OXIDE
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1387 NITRIC OXIDE SYNTHASE
(NITRIC(W) OXIDE(W) SYNTHASE)
10160 HEMATOPO?

L7 205 STEM CELL? AND NITRIC OXIDE SYNTHASE AND HEMATOPO?

=> s 17 and pd<1999

2434829 PD<1999
(PD<199900000)

L8 3 L7 AND PD<1999

=> d 181-3

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 CLMN, DRWN, AB
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 NCLS, EXF, REP, REN, ARTU, EXNAM, LREP, CLMN, DRWN, AB,
 PARN, SUMM, DRWD, DETD, CLM
 FPBIB ----- PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI,
 RLI, PRAI, REP, REN, EXNAM, LREP, CLM, CLMN, DRWN
 FHITSTR ----- HIT RN, its text modification, its CA index name, and
 its structure diagram
 FPG ----- FP plus PAGE.DRAW
 GI ----- PN and page image numbers
 HIT ----- All fields containing hit terms
 HITRN ----- HIT RN and its text modification
 HITSTR ----- HIT RN, its text modification, its CA index name, and
 its structure diagram
 IABS ----- ABS, indented with text labels
 IALL ----- ALL, indented with text labels
 IALLG ----- IALL plus PAGE.DRAW
 IBIB ----- BIB, indented with text labels
 IBIB.EX ----- IBIB for original and latest publication
 IBIBG ----- IBIB plus PAGE.DRAW
 IMAX ----- MAX, indented with text labels
 IMAX.EX ----- IMAX for original and latest publication
 IND ----- INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
 EXF, ARTU, OS, CC, SX, ST, IT
 ISTD ----- STD, indented with text labels
 KWIC ----- All hit terms plus 20 words on either side
 MAX ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
 RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
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 EXF, ARTU OS, CC, SX, ST, IT
 MAX.EX ----- MAX for original and latest publication
 OCC ----- List of display fields containing hit terms
 SBIB ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
 DT, FS, LN.CNT
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 without answer number. SCAN must be entered on the
 same line as DISPLAY, e.g., D SCAN)
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 DT, FS, LN.CNT, INCL, INCLM, INCLS, NCL, NCLM, NCLS,
 IC, ICM, ICS, EXF (STD is the default)
 STD.EX ----- STD for original and latest publication
 TRIAL ----- AN, TI, INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC,
 ICM, ICS

ENTER DISPLAY FORMAT (STD):display format
 'DISPLAY' IS NOT A VALID FORMAT FOR FILE 'USPATFULL'
 'FORMAT' IS NOT A VALID FORMAT FOR FILE 'USPATFULL'

The following are valid formats:

The default display format is STD.

ABS ----- AB
 ALL ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
 RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
 DRWN, AB, GOVI, PARN, SUMM, DRWD, DETD, CLM, INCL,
 INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,

EXF, ARTU

ALLG ----- ALL plus PAGE.DRAW

BIB ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD, RLI, PRAI, DT, FS, EXNAM, LREP, CLMN, ECL, DRWN, LN.CNT

BIB.EX ----- BIB for original and latest publication

BIBG ----- BIB plus PAGE.DRAW

BROWSE ----- See "HELP BROWSE" or "HELP DISPLAY BROWSE". BROWSE must entered on the same line as DISPLAY, e.g., D BROWSE.

CAS ----- OS, CC, SX, ST, IT

CBIB ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PRAI, DT, FS

DALL ----- ALL, delimited for post-processing

FP ----- PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI, RLI, PRAI, IC, ICM, ICS, INCL, INCLM, INCLS, NCL, NCLM, NCLS, EXF, REP, REN, ARTU, EXNAM, LREP, CLMN, DRWN, AB

FP.EX ----- FP for original and latest publication

FPALL ----- PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI, RLI, PRAI, IC, ICM, ICS, INCL, INCLM, INCLS, NCL, NCLM, NCLS, EXF, REP, REN, ARTU, EXNAM, LREP, CLMN, DRWN, AB, PARN, SUMM, DRWD, DETD, CLM

FPBIB ----- PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI, RLI, PRAI, REP, REN, EXNAM, LREP, CLM, CLMN, DRWN

FHITSTR ----- HIT RN, its text modification, its CA index name, and its structure diagram

FPG ----- FP plus PAGE.DRAW

GI ----- PN and page image numbers

HIT ----- All fields containing hit terms

HITRN ----- HIT RN and its text modification

HITSTR ----- HIT RN, its text modification, its CA index name, and its structure diagram

IABS ----- ABS, indented with text labels

IALL ----- ALL, indented with text labels

IALLG ----- IALL plus PAGE.DRAW

IBIB ----- BIB, indented with text labels

IBIB.EX ----- IBIB for original and latest publication

IBIBG ----- IBIB plus PAGE.DRAW

IMAX ----- MAX, indented with text labels

IMAX.EX ----- IMAX for original and latest publication

IND ----- INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS, EXF, ARTU, OS, CC, SX, ST, IT

ISTD ----- STD, indented with text labels

KWIC ----- All hit terms plus 20 words on either side

MAX ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD, RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL, DRWN, AB, GOVI, PARN, SUMM, DRWD, DETD, CLM, INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS, EXF, ARTU OS, CC, SX, ST, IT

MAX.EX ----- MAX for original and latest publication

OCC ----- List of display fields containing hit terms

SBIB ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI, DT, FS, LN.CNT

SCAN ----- AN, TI, NCL, NCLM, NCLS, IC, ICM, ICS (random display without answer number. SCAN must be entered on the same line as DISPLAY, e.g., D SCAN)

STD ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI, DT, FS, LN.CNT, INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS, EXF (STD is the default)

STD.EX ----- STD for original and latest publication

TRIAL ----- AN, TI, INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS

ENTER DISPLAY FORMAT (STD):STD

L8 ANSWER 1 OF 3 USPATFULL
 AN 1998:68519 USPATFULL
 TI Systemic gene treatment of connective tissue diseases with IRAP-1
 IN Evans, Christopher H., Pittsburgh, PA, United States
 Robbins, Paul D., Pittsburgh, PA, United States
 PA University of Pittsburgh of the Commonwealth System of Higher Education,
 Pittsburgh, PA, United States (U.S. corporation)
 PI US 5766585 19980616 <--
 AI US 1996-697180 19960820 (8)
 RLI Continuation of Ser. No. US 1993-167642, filed on 14 Dec 1993, now
 abandoned
 DT Utility
 FS Granted
 LN.CNT 1869
 INCL INCLM: 424/093.210
 INCLS: 424/093.100; 424/093.200; 424/529.000; 424/534.000; 435/172.300;
 435/320.100; 514/044.000; 935/023.000; 935/071.000
 NCL NCLM: 424/093.210
 NCLS: 424/093.100; 424/093.200; 424/529.000; 424/534.000; 435/320.100;
 514/044.000
 IC [6]
 ICM: A01N043-04
 ICS: A01N063-00; C12N005-16; C12N015-07
 EXF 435/69.1; 435/70.1; 435/703; 435/172.1; 435/172.3; 435/320.1; 435/325;
 424/93.1; 424/93.2; 424/93.21; 424/529; 424/534; 514/44; 935/22; 935/32;
 935/34; 935/70; 935/71; 935/23
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> D L8 2-3

L8 ANSWER 2 OF 3 USPATFULL
 AN 97:42862 USPATFULL
 TI Method for producing in vivo delivery of therapeutic agents via
 liposomes
 IN Dzau, Victor J., 12101 Dawn La., Los Altos Hills, CA, United States
 94022
 Kaneda, Yasufumi, Molecular & Cellular Institute, Osaka University, 1-3,
 Yamada-oka, Suita-City, Osaka 565, Japan
 PI US 5631237 19970520 <--
 AI US 1994-241372 19940510 (8)
 RLI Continuation-in-part of Ser. No. US 1992-995022, filed on 22 Dec 1992,
 now abandoned
 DT Utility
 FS Granted
 LN.CNT 2435
 INCL INCLM: 514/044.000
 INCLS: 424/450.000; 424/417.000; 428/402.200; 264/004.100; 264/004.300;
 264/004.600
 NCL NCLM: 514/044.000
 NCLS: 264/004.100; 264/004.300; 264/004.600; 424/417.000; 424/450.000;
 428/402.200
 IC [6]
 ICM: A61K048-00
 ICS: A61K009-127
 EXF 514/44; 514/2; 424/93.1; 424/450; 424/283.1; 424/1.21; 424/1.25;
 435/320.1; 435/69.1; 435/5; 435/193
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 3 OF 3 USPATFULL
 AN 95:58172 USPATFULL

TI Treatment of vascular degenerative diseases by modulation of endogenous
 nitric oxide production of activity
 IN Cooke, John P., Palo Alto, CA, United States
 Dzau, Victor J., Los Altos Hills, CA, United States
 Gibbons, Gary H., Palo Alto, CA, United States
 PA The Board of Trustees of the Leland Stanford Junior University,
 Stanford, CA, United States (U.S. corporation)
 PI US 5428070 19950627 <--
 AI US 1993-76312 19930611 (8)
 DT Utility
 FS Granted
 LN.CNT 683
 INCL INCLM: 514/557.000
 INCLS: 514/310.000
 NCL NCLM: 514/557.000
 NCLS: 514/310.000
 IC [6]
 ICM: A01N037-00
 ICS: A61K031-19
 EXF 514/310; 514/557
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> S HEMATOPOIESIS AND STEM CELLS AND NITRIC OXIDE SYNTHASE
 2016 HEMATOPOIESIS
 126332 STEM
 319013 CELLS
 9569 STEM CELLS
 (STEM(W) CELLS)
 67061 NITRIC
 475839 OXIDE
 9133 SYNTHASE
 1387 NITRIC OXIDE SYNTHASE
 (NITRIC(W) OXIDE(W) SYNTHASE)
 L9 142 HEMATOPOIESIS AND STEM CELLS AND NITRIC OXIDE SYNTHASE

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 2604557 PD< 2000
 (PD<200000000)
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=> D L11 1-2

L11 ANSWER 1 OF 2 USPATFULL
 AN 1999:141294 USPATFULL
 TI Methods for enhancing angiogenesis with endothelial progenitor cells
 IN Isner, Jeffrey M., Weston, MA, United States
 Asahara, Takayuki, Arlington, MA, United States
 PA St. Elizabeth's Medical Center of Boston, Boston, MA, United States
 (U.S. corporation)
 PI US 5980887 19991109 <--
 AI US 1996-744882 19961108 (8)
 DT Utility
 FS Granted
 LN.CNT 1104
 INCL INCLM: 424/093.700
 INCLS: 424/085.100; 424/085.200; 514/008.000; 514/044.000

NCL NCLM: 424/093.700
NCLS: 424/085.100; 424/085.200; 514/008.000; 514/044.000
IC [6]
ICM: A61K035-12
ICS: A61K048-00; A61K038-18; A61K038-19
EXF 424/93.7; 424/85.4; 424/85.2; 435/325; 435/375; 514/2; 514/8; 514/44;
530/351; 053/23.5
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 2 OF 2 USPATFULL
AN 1999:137330 USPATFULL
TI Therapeutic uses for nitric oxide inhibitors
IN Enikolopov, Grigori N., Cold Spring Harbor, NY, United States
Peunova, Natalia I., Cold Spring Harbor, NY, United States
Kuzin, Boris A., Moscow, Russian Federation
Michurina, Tatiyana, Moscow, Russian Federation
PA Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, United States
(U.S. corporation)
PI US 5977181 19991102 <--
AI US 1997-969475 19971113 (8)
PRAI US 1996-30690P 19961113 (60)
US 1997-45411P 19970502 (60)
DT Utility
FS Granted
LN.CNT 1655
INCL INCLM: 514/631.000
INCLS: 514/565.000; 514/632.000
NCL NCLM: 514/631.000
NCLS: 514/565.000; 514/632.000
IC [6]
ICM: A61K031-195
ICS: A61K031-155
EXF 514/565; 514/631; 514/632
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> D L11 1-2 KWIC

L11 ANSWER 1 OF 2 USPATFULL
PI US 5980887 19991109 <--
DETD . . . typically begins as a cluster formation, or blood island, comprised of EC progenitors (e.g. angioblasts) at the periphery and hematopoietic **stem cells** (HSCs) at the center (3).
In addition to this intimate and predictable spatial association, such EC progenitors and HSCs share. . .
DETD The demonstration that transplants of HSCs derived from peripheral blood can provide sustained hematopoietic recovery constitutes inferential evidence for circulating **stem cells**. (5). This observation is now being exploited clinically as an alternative to bone marrow transplantation.
DETD . . . genes or their encoded gene products to enhance the activity of targeted cells, while simultaneously inducing angiogenesis, including, for example, **nitric oxide synthase**, L-arginine, fibronectin, urokinase, plasminogen activator and heparin.
DETD ECs uniquely express endothelial constitutive **nitric oxide synthase** (ecNOS). Accordingly, MB.sup.CD34+, MB.sup.CD34- and AT.sup.CD34+ were investigated for expression of ecNOS by RT-PCR (15). ecNOS mRNA was not detectable. . .
DETD Cell-cell interaction is considered to play a decisive role in cell signaling, differentiation, and proliferation during **hematopoiesis** (19) and angiogenesis (20). To study the impact of MB.sup.CD34+ interaction with mature ECs on the differentiation of

L11 ANSWER 2 OF 2 USPATFULL

PI US 5977181

19991102

<--

AB . . . developing organism. In particular, the present invention relates to a method of increasing in a mammal a population of hematopoietic **stem cells** in bone marrow which are capable of undergoing normal **hematopoiesis** and differentiation, wherein the bone marrow is contacted with an inhibitor of NO, such as an inhibitor of **nitric oxide synthase** (NOS), thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation. The present invention also relates to a method of increasing a population of cells in S phase in. . .

SUMM . . . developing organism. In particular, the present invention relates to a method of increasing in a mammal a population of hematopoietic **stem cells**, including precursors to myeloid, lymphoid and erythroid cells, in bone marrow which are capable of undergoing normal **hematopoiesis** and differentiation, wherein the bone marrow is contacted with an inhibitor of NO, such as an inhibitor of **nitric oxide synthase** (NOS), thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation. The method can be carried out in vivo or ex vivo. In addition, the method can be used. . .

SUMM The present invention also relates to a method for treating a mammal to increase a population of hematopoietic **stem cells** in bone marrow of the mammal which are capable of undergoing normal **hematopoiesis** and differentiation. In the method, the bone marrow of the mammal is contacted with an inhibitor of NOS, thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation. The method can further comprise contacting the bone marrow with at least one agent which induces differentiation of. . .

SUMM In one embodiment of the method for treating a mammal to increase a population of hematopoietic **stem cells** in bone marrow of the mammal which are capable of undergoing normal **hematopoiesis** and differentiation, bone marrow which is to be transplanted is obtained, wherein the bone marrow to be transplanted can be. . . is transplanted into the mammal being treated, thereby providing the mammal with bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation. The method can further comprise treating the mammal with an inhibitor of NOS before or after transplanting the. . .

SUMM . . . method, bone marrow is contacted with an inhibitor of NOS, thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation; and at least one agent (e.g., a hematopoietic growth factor) selected to induce specific differentiation of the hematopoietic. . .

SUMM Accordingly, the present invention relates to a method of increasing in a mammal a population of hematopoietic **stem cells**, including precursors to myeloid, lymphoid and erythroid cells, in bone marrow which are capable of undergoing normal **hematopoiesis** and differentiation, by contacting the bone marrow with an inhibitor of NO, such as an inhibitor of NOS. The present invention includes a method for treating a mammal to increase a population of hematopoietic **stem cells** in bone marrow of the mammal which are

capable of undergoing normal **hematopoiesis** and differentiation, in which the bone marrow of the mammal is contacted with an inhibitor of NOS.

SUMM . . . lymphocytes, neutrophils and platelets). For example, in the embodiment wherein a mammal is treated to increase a population of hematopoietic **stem cells** in the bone marrow of the mammal by contacting the bone marrow of the mammal with an inhibitor of NOS, . . .

SUMM . . . balance between cell proliferation and cell differentiation. Moreover, results shown here demonstrate that NO acts as a crucial regulator of **hematopoiesis** after bone marrow (BM) transplantation. NO regulates the maturation of both the erythroid and myeloid lineages. These data demonstrate that manipulations of NOS activity and NO levels during **hematopoiesis** can be used to alter (enhance or reduce) blood cell production. This is useful for preventive and therapeutic intervention.

SUMM As also described herein, the role of NO in **hematopoiesis** was examined. To demonstrate the presence of NOS in the bone marrow (BM) cells, BM from adult mice was tested. . .

SUMM A mouse model of syngeneic BM transfer was used to evaluate the role of NO in **hematopoiesis**. Mice were irradiated to inhibit **hematopoiesis** in the recipient animal, BM was transplanted from syngeneic animals, and the animals were treated with specific NOS inhibitors. This procedure permits the proliferation, differentiation and survival of only the transplanted cells. To study the changes in **hematopoiesis** introduced by NOS inhibitors, the colonies in the spleen were monitored to test the differentiation of erythroid cells, and the . . . of the recipients were monitored to test the differentiation of cells of the granulocyte-macrophage lineage. The role of NO on **hematopoiesis** was tested by injecting the animals with the specific and structurally unrelated NOS inhibitors L-nitroarginine methyl ester (L-NAME), and 2-ethyl-2-thiopseudourea. .

SUMM Taken together, the results of these studies indicate that NO modulates **hematopoiesis** after BM transplantation. This confirms the role of NO as a major regulatory factor in the organism controlling the balance. . .

SUMM The results of work described herein support the ability of NO to act as a crucial regulator of **hematopoiesis** after bone marrow transplantation (BMT). NO regulates maturation of both erythroid and myeloid cell lineages. By interfering with NO production. . . and 20-fold for the erythroid lineage. The data described herein demonstrates that manipulations of NOS activity and NO levels during **hematopoiesis** can be used for therapeutic purposes to influence self renewal and differentiation of hematopoietic **stem cells**, and to replace damaged or defective cells. Areas of application include enhancement of blood cell and myeloid cell formation following. . .

SUMM . . . chondrocyte differentiation. These results show that manipulation of NO production can regulate growth and differentiation of osteoblasts, chondrocytes, or mesenchymal **stem cells**. This can be used for amplification and further differentiation of cells in the injured tissue, or for cell implants (in. . .

DETD Nitric Oxide Regulates **Hematopoiesis** in Animals Erythroid Differentiation

DETD . . . cGy total body irradiation within 3-4 hours before transplantation. This dosage was tested to be enough for complete suppression of **hematopoiesis** in the irradiated recipient animals. BM cells were flushed from the femurs of syngeneic donors and injected intravenously (10.sup.5 BM. . .

DETD . . . granulocyte colony stimulating factor (G-CSF-R) and erythropoietin (EpoR). The appearance of each of these receptors marks a

specific stage in **hematopoiesis**.

DETD **Stem Cells** in the Bone Marrow

DETD . . . the presence of various growth factor receptors which serve as markers of the differentiation stage and indicate the presence of **stem cells** and multipotent precursor cells. The BM preparations were tested for cells expressing receptors to HSF (ligand of c-kit), GM-CSF, G-CSF. . . number of c-kit-positive and IL-3-R-positive cells, suggesting that the population of cells in the BM becomes highly enriched in hematopoietic **stem cells**. At the same time the number of cells expressing receptors for G-CSF, which marks the later stages of differentiation, decreases almost three-fold, while the number of GM-CSF-R-positive cells is slightly decreased. This suggests that inhibition of NOS during **hematopoiesis** selectively enriches the BM in undifferentiated **stem cells** which have already acquired c-kit and IL3 receptors, but have not proceeded to the later stages when the receptor for. . .

DETD The critical question is whether undifferentiated **stem cells** which accumulate in the bone marrow as a result of treatment with NOS inhibitors have the capacity to revert to normal state and resume normal **hematopoiesis** process once the action of NOS inhibitors is suspended. The failure to do so might indicate that the cells become. . . with NOS inhibitors was halted 7-9 days after the BM transfer and checked the BM cells for the presence of **hematopoiesis** markers 1-7 days after termination of injections. Control mice continued to receive the daily injections, The results (Table 4) demonstrate. . . cells were able to resume their differentiation and to proceed to the later stages normally. This indicates that enrichment in **stem cells** after treatment with NOS inhibitors is reversible and can be used to "boost" the number of **stem cells** before inducing them to proceed further along their differentiation pathways.

DETD . . . as a result, regulates the balance between cell proliferation and differentiation in cultured neuronal cells, in developing Drosophila, and during **hematopoiesis** in mammals (Peunova et al., 1996; Kuzin et al., 1996; Michurina et al., 1997). Here, whether NOS is involved in. . .

CLM What is claimed is:

1. A method of increasing in a mammal a population of hematopoietic **stem cells** in bone marrow which are capable of undergoing normal **hematopoiesis** and differentiation, comprising contacting the bone marrow with an inhibitor of **nitric oxide synthase**, thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation.

3. A method according to claim 2 further comprising implanting the bone marrow having an increased population of hematopoietic **stem cells** into a mammal in need thereof.

7. A method according to claim 1 wherein the inhibitor of **nitric oxide synthase** is selected from the group consisting of L-nitroarginine methyl ester, 2-ethyl-2-thiopseudourea, aminoguanidine hemisulfate and N-monomethyl-L-arginine.

8. A method for treating a mammal to increase a population of hematopoietic **stem cells** in bone marrow of the mammal which are capable of undergoing normal **hematopoiesis** and differentiation, comprising contacting the bone marrow of the mammal with an inhibitor of **nitric oxide synthase**, thereby producing bone marrow having an increased population of

hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation.

10. A method for treating a mammal to increase a population of hematopoietic **stem cells** in bone marrow of the mammal which are capable of undergoing normal **hematopoiesis** and differentiation, comprising the steps of: a) obtaining bone marrow which is to be transplanted into the mammal; b) contacting the bone marrow to be transplanted with an inhibitor of **nitric oxide synthase**; c) transplanting the bone marrow of step (b) into the mammal to be treated, thereby providing the mammal with bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation.

11. A method according to claim 10 further comprising: d) treating the mammal with an enhancer of **nitric oxide synthase** after transplanting the bone marrow.

12. A method according to claim 10 further comprising: d) treating the mammal with an inhibitor of **nitric oxide synthase** after transplanting the bone marrow.

. . . method of producing a subpopulation of hematopoietic cells comprising the steps of: a) contacting bone marrow with an inhibitor of **nitric oxide synthase**, thereby producing bone marrow having an increased population of hematopoietic **stem cells** which are capable of undergoing normal **hematopoiesis** and differentiation; and b) contacting the bone marrow with at least one hematopoietic growth factor selected to induce specific differentiation. . . .